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REVIEW

Gut-liver axis in diabetes: Mechanisms and therapeutic opportunities

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Abstract

The gut-liver axis represents a complex, bidirectional communication network between the gastrointestinal tract and the liver, playing a central role in maintaining metabolic homeostasis. In diabetes, disruption of this axis, mediated by gut microbiota dysbiosis, impaired intestinal barrier function, and pro-inflammatory signaling, contributes significantly to insulin resistance, hepatic steatosis, and systemic metabolic dysfunction. This review explores the underlying mechanisms by which microbial alterations, increased gut permeability, and inflammatory pathways influence hepatic insulin resistance and glucose metabolism. In addition to established mechanisms, emerging pathways involving neuroendocrine circuits, microbial metabolites, and immune mediators are discussed, offering deeper insight into gut-liver interactions in metabolic disease. The review also outlines therapeutic strategies targeting the gut-liver axis, including microbiota modulation, barrier function enhancement, and anti-inflammatory interventions, emphasizing their potential in advancing diabetes management. A conceptual framework is proposed to integrate these components into a precision medicine approach for metabolic regulation. Key challenges in clinical translation, including patient heterogeneity and the absence of reliable biomarkers to guide treatment decisions are also discussed to inform future research. By linking mechanistic understanding with therapeutic innovation, the review highlights the gut-liver axis as a promising target for personalized diabetes care.

Key Words: Diabetes; Microbiota; Diabetes management; Gut-liver axis; Anti-inflammatory; Intestinal permeability; Metabolic homeostasis

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Core Tip: Disruption of the gut-liver axis plays a critical role in the pathophysiology of diabetes by linking gut microbiota alterations, intestinal barrier dysfunction, and inflammatory pathways to hepatic insulin resistance and impaired glucose metabolism. Understanding these mechanisms opens new therapeutic opportunities, including microbiota modulation, gut barrier reinforcement, and targeted anti-inflammatory strategies. These approaches have the potential to significantly improve diabetes management and metabolic outcomes.

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INTRODUCTION

Diabetes mellitus is a complex metabolic disorder characterized by chronic hyperglycemia, insulin resistance (IR), and progressive β-cell dysfunction[1]. Over the past decade, growing evidence has established the gut-liver axis as a pivotal contributor to diabetes pathogenesis, offering novel insights into disease mechanisms and therapeutic opportunities. The gut-liver axis forms an integrated communication network linking metabolic, immune, and endocrine signals between the gastrointestinal tract and the liver[2]. This dynamic crosstalk is mediated by gut microbiota composition, intestinal barrier integrity, microbial metabolites, and inflammatory signaling pathways[3-5]. Disruption of this system contributes to IR, hepatic dysfunction, and systemic metabolic dysregulation, core features of diabetes[5].

Globally, the burden of diabetes has reached alarming levels, with an estimated 828 million adults affected in 2022, an increase of more than 630 million cases since 1990. This surge is closely linked to lifestyle changes, including increased consumption of ultra-processed foods, reduced physical activity, and escalating obesity rates, all of which also influence gut microbiota composition and liver health[6].

Non-alcoholic fatty liver disease (NAFLD), recently redefined as metabolic dysfunction-associated steatotic liver disease (MASLD), has emerged as a key comorbidity of type 2 diabetes (T2D). A meta-analysis of nearly 7 million individuals with NAFLD and over 1 million with MAFLD found that the pooled global prevalence of T2D among these populations was 28.3% and 26.2%, respectively, with incidence densities of 24.6 and 26.9 cases *per* 1000 person-years[7]. These findings confirm that liver fat accumulation is not only a consequence of metabolic dysfunction but also a strong predictor of diabetes onset. Conversely, diabetes accelerates the progression of steatotic liver disease to more advanced stages, including non-alcoholic steatohepatitis, fibrosis, and cirrhosis. This bidirectional relationship between liver disease and diabetes underscores the pivotal role of the gut-liver axis in metabolic disease and highlights the need for integrative treatment strategies targeting both organs.

Gut dysbiosis, an imbalance in microbial composition and function, has been implicated as a central factor in diabetes pathophysiology[8,9]. Individuals with diabetes often exhibit altered microbial profiles, increased intestinal permeability ("leaky gut"), systemic inflammation, and hepatic IR[10,11]. Moreover, disruptions in microbial metabolites such as short-chain fatty acids (SCFAs) and bile acid derivatives further impair metabolic function[12,13], positioning gut dysbiosis as a promising therapeutic strategy.

While most studies focus on T2D, emerging data also implicate gut-liver axis disruptions in type 1 diabetes (T1D) and type 3 diabetes (T3D). In T1D, an autoimmune condition involving pancreatic β-cell destruction, gut microbiota alterations, and increased gut permeability may contribute to immune dysregulation[14-16]. A loss of butyrate-producing bacteria, critical for maintaining immune tolerance, is commonly observed in individuals with T1D[17,18]. Similarly, in T3D, a term used to describe IR and metabolic dysfunction associated with Alzheimer's disease, gut-liver axis disruptions have been linked to cognitive decline[19-21]. Hepatic IR and MASLD are recognized risk factors for neurodegeneration, with gut-derived inflammation implicated in promoting amyloid-beta accumulation and tau hyperphosphorylation[22-25].

Thus, the gut-liver axis stands at the crossroads of metabolic and neurodegenerative diseases, offering a unifying framework for understanding complex disease interactions. In the context of diabetes, unravelling this axis could unlock innovative therapies to restore metabolic homeostasis. Building on this perspective, the present review provides a comprehensive analysis of the gut-liver axis in diabetes, focusing on its mechanistic foundations and therapeutic relevance. Specifically, the objectives are to: (1) Describe the molecular and cellular mechanisms connecting gut microbiota, intestinal permeability, and hepatic metabolism; (2) Highlight emerging pathways involving neuroendocrine, immune, and microbial signaling; (3) Evaluate current and potential interventions targeting these mechanisms; and (4) Identify key knowledge gaps and translational challenges. Through this integrative approach, the review aims to offer a framework to inform future research and support more effective strategies for diabetes care.

THE GUT-LIVER AXIS: PHYSIOLOGICAL OVERVIEW

The gut-liver axis is a highly coordinated, bidirectional communication system that plays a critical role in maintaining metabolic equilibrium, immune regulation, and nutrient processing[5,26]. This connection is anatomically and

functionally centered on the portal vein, which delivers nutrient- rich and microbe-derived blood directly from the intestine to the liver[27]. As a result, the liver continuously samples intestinal inputs, serving as both a metabolic hub and immune sentinel[28].

At the core of this axis lies the intestinal barrier, a multilayered defense system composed of mucus, epithelial cells joined by tight junctions, and mucosal immune components[29]. Its primary function is to allow nutrient absorption while preventing the translocation of pathogens and toxins into the bloodstream. Disruption of this barrier, often due to inflammation, poor diet, or dysbiosis, allows microbial products such as lipopolysaccharides (LPS) to enter the portal circulation, triggering hepatic immune responses that promote inflammation and metabolic dysfunction[29-32].

The gut microbiota, a diverse ecosystem of trillions of microorganisms, plays a central role in modulating gut-liver communication[3,8,33]. Microbial fermentation of dietary fibers produces SCFAs, including butyrate, acetate, and propionate, which serve as energy sources for colonocytes and influence both intestinal and hepatic immune function[34, 35]. SCFAs also regulate gluconeogenesis, lipid metabolism, and insulin sensitivity *via* G-protein-coupled receptors and through epigenetic modifications such as histone acetylation[36-38].

Bile acids, synthesized in the liver and modified by gut microbiota, also act as key signaling molecules[39,40]. Secondary bile acids generated by microbial metabolism are reabsorbed and recirculated back to the liver *via* the entero-hepatic circulation[41,42]. These bile acids activate nuclear receptors such as the farnesoid X receptor (FXR) and Takeda G-protein-coupled receptor 5 (TGR5), thereby modulating lipid metabolism, glucose homeostasis, and inflammatory signaling pathways[43,44].

Additionally, gut microbes convert dietary choline and carnitine into trimethylamine-N-oxide (TMAO), a metabolite associated with IR, hepatic steatosis, and atherosclerosis. Elevated TMAO levels exacerbate oxidative stress and alter cholesterol metabolism, further linking gut microbial activity to liver dysfunction[45-47]. A visual representation of these physiological processes is provided in Figure 1.

Together, the intestinal barrier, gut microbiota, microbial metabolites, and hepatic receptors form a dynamic network essential for maintaining systemic metabolic health. Disruption of this network impairs insulin signaling, promotes hepatic steatosis, and contributes to the development of T2D.

Building on this physiological framework, recent studies have identified specific molecular mediators and microbial signatures that mechanistically link gut-liver axis disruption to IR and T2D. The next section outlines these key mediators, including the roles of immune activation, receptor signaling, and microbial metabolites driving metabolic dysfunction.

KEY MEDIATORS OF THE GUT–LIVER AXIS IN DIABETES AND IR

Pro-inflammatory cytokines and immune signaling

A hallmark of gut-liver axis disruption in T2D is the upregulation of inflammatory signaling. Pro-inflammatory signaling. Elevated levels of pro-inflammatory cytokines, including interleukin-6, tumor necrosis factor-α, and interleukin-1beta (IL-1β) are consistently observed in insulin-resistant states. These cytokines impair insulin signaling by inhibiting the phosphorylation of insulin receptor substrate-1 (IRS-1), thereby disrupting downstream phosphatidylinositol 3-kinase-protein kinase B pathways[48-51].

A key driver of this inflammation is LPS, an endotoxin produced by Gram-negative gut bacteria. When intestinal barrier integrity is compromised, LPS translocates into the portal circulation and binds to Toll-like receptor 4 (TLR4) on hepatic Kupffer cells. This interaction activates the nuclear factor-kappa B (NF-κB) and c-Jun N-terminal kinase (JNK) pathways, promoting hepatic inflammation and worsening IR[29,52-57]. The TLR4–JNK–NF-κB axis thus forms a critical molecular bridge between microbial dysbiosis and hepatic metabolic dysfunction[58-62], as depicted in Figure 2.

Bile acid-activated receptors: FXR and TGR5

Bile acid signaling further exemplifies this interplay. The nuclear receptor FXR and the membrane-bound receptor TGR5 are activated by bile acids and exert dual metabolic and anti-inflammatory roles. FXR activation suppresses hepatic gluconeogenesis and lipogenesis while attenuating NF-κB- mediated inflammation. In the intestine, FXR induces fibroblast growth factor (FGF) 19, which acts hormonally to inhibit hepatic glucose production. Similarly, TGR5 activation on intestinal L-cells promotes glucagon-like peptide-1 (GLP-1) secretion and suppresses hepatic inflammatory responses. Dysbiosis-induced alterations in bile acid pools can attenuate FXR/TGR5 signaling, further worsening metabolic dysregulation[63-65].

Microbial dysbiosis

The composition and function of gut microbiota are essential for maintaining intestinal barrier integrity and regulating host metabolism. Individuals with T2D often exhibit reduced microbial diversity and depletion of beneficial commensals such as *Faecalibacterium prausnitzii*, *Roseburia intestinalis*, and *Akkermansia muciniphila*. These microbes are key producers of SCFAs, which help preserve epithelial integrity, regulate immune responses, and enhance insulin sensitivity. Their loss compromises tight junctions and mucin layers, increasing intestinal permeability and promoting a pro-inflammatory gut environment[66-69].

Concurrently, pathobionts such as *Escherichia coli*, *Prevotella copri*, and *Bacteroides vulgatus* become more prevalent in T2D. These microbes produce LPS, contributing to elevated systemic LPS levels and inducing metabolic endotoxemia[8, 70-72]. Notably, *Prevotella copri* is associated with increased production of branched-chain amino acids (BCAAs), particularly leucine and valine. These BCAAs activate the mechanistic target of rapamycin-ribosomal protein S6 kinase 1 signaling pathway, which phosphorylates IRS-1 at inhibitory sites and disrupts of insulin signaling in the liver and

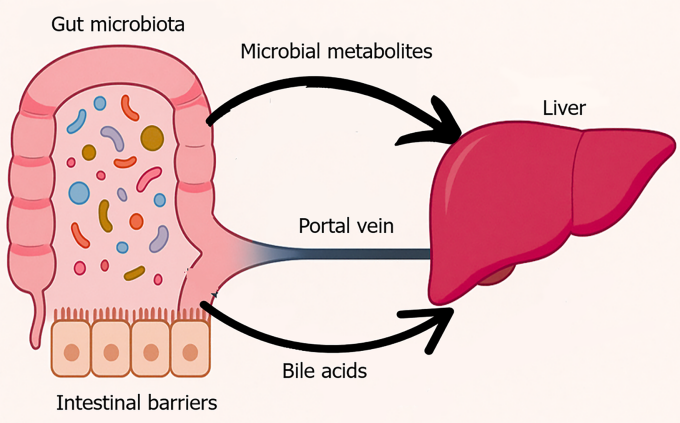

Figure 1 Physiological gut liver axis. This figure visually represents the anatomical and functional connectivity between the gut and liver, commonly referred to as the gut-liver axis. In a healthy state, a diverse and balanced gut microbiota resides within the intestinal lumen, supported by an intact epithelial barrier. Microbial metabolites such as short-chain fatty acids and secondary bile acids enter the portal circulation and interact with the liver to regulate glucose and lipid metabolism, immune tolerance, and bile acid recycling. This bidirectional communication maintains metabolic and immune homeostasis.

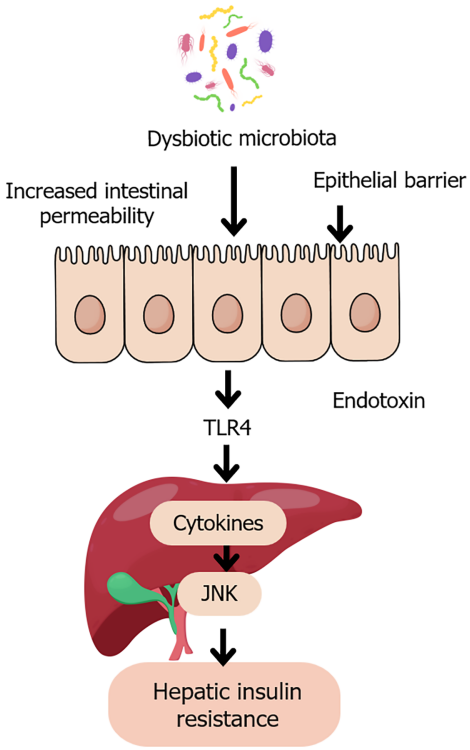

Figure 2 Gut dysbiosis and hepatic insulin resistance. This figure is a schematic illustration of the pathological pathway linking dysbiotic gut microbiota to hepatic insulin resistance. Disruption of microbial balance leads to increased intestinal permeability and epithelial barrier dysfunction, allowing the translocation of endotoxins such as lipopolysaccharide into the portal circulation. These endotoxins activate toll-like receptor 4 on hepatic Kupffer cells, triggering pro-inflammatory cytokine release and activation of c-Jun N-terminal kinase signaling. This inflammatory cascade impairs insulin receptor signaling, contributing to hepatic insulin resistance, a key feature of type 2 diabetes. JNK: C-Jun N-terminal kinase; TLR4: Toll-like receptor 4.

muscle[73,74].

In addition, *Akkermansia muciniphila*, a mucin- degrading bacterium vital for tight junction function is often depleted in T2D. Its absence is linked to increased gut permeability and liver inflammation[75,76]. This enhanced permeability enables microbial products such as LPS to enter the portal circulation, activating hepatic immune responses and worsening systemic IR[11,77]. Restoration of *Akkermansia muciniphila* through prebiotics or supplementation has shown promise in improving metabolic parameters in both preclinical and early clinical studies[78,79].

Microbial and host-derived metabolites

Several gut and host-derived metabolites act as key modulators of gut-liver axis regulation and contribute to IR in T2D. Among them, LPS produced by Gram-negative bacteria, remains a central trigger of endotoxin-driven inflammation by

activating hepatic immune pathways[54,59,60]. Secondary bile acids, formed through microbial conversion of primary bile acids, modulate FXR and TGR5 signaling. Altered bile salt hydrolase activity and disrupted bile acid profiles impair receptor activation, contributing to glucose dysregulation and hepatic lipotoxicity[39,43,64,65].

Another key metabolite is imidazole propionate (ImP), derived from microbial histidine metabolism. ImP interferes with insulin signaling *via* activation of p38γ and mechanistic target of rapamycin complex 1, and its elevated plasma levels have been linked to both prediabetes and T2D, suggesting its value as a biomarker and therapeutic target[80-82].

Tryptophan-derived indole metabolites, including indole-3-propionic acid and 5-hydroxyindole-3-acetic acid (5-HIAA), interact with the aryl hydrocarbon receptor (AhR) to modulate hepatic inflammation and insulin sensitivity. Reduced levels of these metabolites are associated with impaired glucose tolerance and elevated systemic inflammation in T2D[83].

Other significant microbial metabolites include phenylacetylglutamine, a co-metabolite of dietary phenylalanine that activates adrenergic receptors and promotes systemic inflammation[84]; hydrogen sulfide, which exhibits anti-inflammatory properties at low concentrations but becomes cytotoxic at higher levels, impairing mitochondrial function and compromising intestinal integrity[85]; and microbial succinate, a metabolite that signals through *SUCNR1* to drive hepatic fibrosis and inflammation[86].

Additionally, endogenous ethanol (EE), produced by microbes such as *Klebsiella pneumoniae* and *Candida spp.*, contributes to oxidative stress, hepatic steatosis, and mitochondrial dysfunction. Elevated EE levels have been observed in individuals with MASLD even in the absence of alcohol intake, identifying EE as a gut-derived metabolic toxin in diabetes[87-89].

A particularly novel mechanism involves bacterial extracellular vesicles (BEVs), nano-sized vesicles secreted by both commensal and pathogenic bacteria. BEVs can cross intact epithelial barriers and deliver microbial molecules, such as LPS and DNA, directly to hepatic immune cells. This enables microbial communication with the liver even without overt barrier disruption, providing a distinct pathway for inducing hepatic inflammation and IR[90,91].

Secretory immunoglobulin A and mucosal immune tolerance

Secretory immunoglobulin A (sIgA) plays a vital role in maintaining mucosal immune homeostasis and intestinal barrier integrity. It neutralizes pathogens, prevents their adherence to epithelial surfaces, and promotes a tolerogenic immune environment by modulating antigen presentation and dendritic cell function[92-94]. In T2D, impaired sIgA production or transport has been linked to reduced microbial diversity, enrichment of pro-inflammatory taxa, and increased gut permeability[92,95,96]. Experimental models show that sIgA deficiency facilitates microbial antigen translocation across the epithelium, triggering hepatic Kupffer cell activation and metabolic inflammation[97,98]. Clinically, decreased sIgA levels are observed in individuals with metabolic syndrome and correlate with markers of IR and systemic inflammation, underscoring sIgA's role in gut-liver immune crosstalk[99].

Paneth and goblet cell dysfunction

Paneth and goblet cells are specialized intestinal epithelial cells essential for maintaining gut barrier integrity through the secretion of antimicrobial peptides and mucins, respectively[100,101]. In diabetic states, both cell types exhibit functional impairments[15]. Rodent models of IR show reduced expression of mucin and antimicrobial peptide genes, thinning of the mucus layer, and architectural changes in intestinal crypts[102]. In humans with diabetes, downregulation of transcripts such as *MUC2* and *DEFA5* indicates secretory epithelial exhaustion[15]. Interestingly, some studies report increased Paneth cell numbers in early diabetes, albeit with defective granules, suggesting a potential compensatory or biphasic response[103]. These findings highlight the need for stage-specific profiling of epithelial alterations. Given that dysfunction of these secretory cells facilitates microbial encroachment and antigen translocation, their impairment may act as an early trigger for gut-liver immune activation and metabolic disruption.

Circadian rhythm disruption

Circadian rhythm disruption is increasingly recognized as a systemic stressor affecting both gut and liver physiology. Intestinal epithelial clock genes regulate tight junction expression, epithelial turnover, and nutrient absorption in a time-dependent manner[104]. Experimental models show that circadian misalignment, induced by sleep fragmentation or altered light-dark cycle, can reshape the gut microbiome, impairs barrier integrity, and increases susceptibility to metabolic endotoxemia[105]. Concurrent disruption of hepatic circadian rhythms impairs bile acid metabolism and glucose homeostasis, contributing to steatosis and IR[106]. However, these effects are not strictly unidirectional. Time-restricted feeding has shown partial restoration of metabolic function even under circadian disruption, and not all core clock gene deletions result in metabolic liver disease[107]. These complexities suggest that circadian misalignment may amplify pre-existing gut-liver axis dysfunction in diabetes rather than initiate it independently.

Autophagy and epithelial stress

Autophagy, a cellular degradation and recycling process, is essential for maintaining epithelial resilience and barrier function. Secretory epithelial cells, including goblet and Paneth cells, depend on intact autophagy pathways to support granule secretion and mitigate endoplasmic reticulum stress[108]. In diabetes, persistent metabolic stress impairs autophagic flux, leading to epithelial apoptosis, mucus depletion, and tight junction disruption. Experimental studies support this link, showing that high-glucose conditions in intestinal organoids elevate oxidative stress and promote barrier breakdown, while interventions enhancing autophagy, such as adenosine 5'-monophosphate-activated protein kinase activators, restore barrier proteins and reduce inflammation[109]. Nonetheless, human data remain limited, and it is unclear whether autophagy disruption is a direct consequence of IR or a secondary effect of dysbiosis and inflam-

mation[110]. Despite these uncertainties, autophagy appears to serve as a key molecular checkpoint for epithelial stress adaptation, and its impairment likely contributes to the breakdown of gut homeostasis and hepatic inflammation observed in T2D[111].

These interconnected mechanisms reflect the multifaceted disruption of the gut-liver axis in diabetes. Table 1 and Figure 3 provide an integrated overview of these pathways.

EMERGING MECHANISMS LINKING THE GUT–LIVER AXIS TO DIABETES

Recent studies have identified novel gut-liver communication pathways that contribute to the pathophysiology of T2D. Three principal axes emerged, involving distinct signals modes, neural, hormonal, and microbial that link gut sensing to hepatic and systemic metabolism.

Gut-liver-brain neuroendocrine crosstalk

This pathway involves gut derived hormones regulating hepatic glucose homeostasis *via* vagal nerve signaling. GLP-1, secreted by intestinal L-cells in response to nutrient intake, stimulates vagal afferents projecting to the hindbrain. This enhances postprandial insulin secretion and promotes satiety. However, obesity and high-fat diets induce an "enteric GLP-1 resistance", characterized by reduced nitric oxide signaling and impaired vagal activation. This blunts the gut-brain insulinotropic axis, contributing to hyperglycemia and hepatic IR in T2D[112].

Similarly, FGF19, secreted by ileal cells in response to bile acid-FXR activation, acts on the hindbrain to suppress hepatic glucose production[113]. Impaired FGF19 signaling, along with GLP-1 resistance, exacerbates dysfunction in this axis[114,115].

FGF21, predominantly synthesized by the liver, further bridges the gut-liver-adipose axis. Its expression is regulated by bile acid-FXR signaling. Pharmacologic induction of FGF21 suppresses hepatic *CYP7A1* expression, alters bile acid composition, and supports expansion of SCFA-producing gut microbiota[116]. These shifts enhance GLP-1 secretion, strengthen gut barrier function, and improve insulin sensitivity. FGF21 also stimulates adiponectin release, thermogenesis, and lipid clearance in peripheral tissues, thereby improving glucose homeostasis[117]. Clinical trials evaluating FGF21 analogs have demonstrated promising reductions in liver fat and glycemic parameters, underscoring its therapeutic relevance[118,119].

Tryptophan-AhR axis

Emerging evidence highlights the tryptophan-AhR axis as a key microbial-host signaling mechanism in T2D. Gut commensals metabolize dietary tryptophan into indole derivatives, including indole-3-acetic acid, indole-3-aldehyde, and 5-HIAA, which act as natural ligands for the AhR. Activation of AhR in hepatic macrophages and hepatocytes exerts anti-inflammatory and metabolic effects by suppressing NF-κB and *STAT1* signaling and enhancing the IL-10-*STAT3* axis [120]. Moreover, AhR activation also downregulates hepatic lipogenic pathways through inhibition of sterol regulatory element-binding protein 1c and its downstream targets, such as acetyl-CoA carboxylase and fatty acid synthase[120].

Notably, supplementation with 5-HIAA has been shown to ameliorate glucose intolerance and hepatic IR in experimental models of obesity. This effect is mediated through AhR-dependent upregulation of *TSC2* and subsequent suppression of mechanistic target of rapamycin complex 1[121]. Collectively, these findings position the gut microbial-AhR axis as a critical modulator of hepatic inflammation and glucose metabolic in T2D[121].

Innate lymphoid cells and endocannabinoid signaling

Beyond systemic hormonal and microbial pathways, recent findings implicate additional immune and metabolic regulators in gut-liver axis dysfunction, particularly innate lymphoid cells (ILCs) and the endocannabinoid system (ECS). These pathways add further complexity to the immune-metabolic interplay between the intestine and liver in T2D[122-126].

ILCs: ILCs are a heterogeneous group of immune cells primarily located at mucosal surfaces, including the intestinal lamina propria, where they contribute to barrier integrity, immune surveillance, and tissue repair[122]. Among these, group 3 ILCs (ILC3s) are particularly important in gut-liver communication. ILC3s secrete interleukin-22 (IL-22), a cytokine that supports epithelial tight junction integrity, stimulates antimicrobial peptide production, and facilitates mucosal healing[127,128].

In metabolic disease models, reduced IL-22 production correlates with increased intestinal permeability, endotoxemia, hepatic inflammation, and IR[[129,130]. Conversely, therapeutic augmentation of IL-22 signaling has been to shown to restore intestinal barrier integrity, decrease portal LPS translocation, and improve hepatic steatosis and insulin sensitivity [131,132]. These findings position ILC3-derived IL-22 as a promising target for restoring gut–liver axis homeostasis in diabetes.

ECS: The ECS consists of cannabinoid receptors (CB1 and CB2), endogenous ligands such as anandamide, 2-arachidonoylglycerol, and associated metabolic enzymes. ECS plays a significant role in gut-liver communication by regulating intestinal permeability, immune responses, microbiota composition, and hepatic metabolism[126].

In diabetes and obesity, CB1 hyperactivation in the gut and liver contributes to dysbiosis, barrier dysfunction, adipogenesis, hepatic steatosis, and IR[133,134]. Pharmacologic blockade of CB1 receptors has been shown to reverse these effects, restoring gut barrier function and improving hepatic insulin sensitivity in animal models[135,136].

Table 1 Key mediators of the gut-liver axis relevant to diabetes and insulin resistance

Mediator	Mechanism of action	Clinical relevance in T2D	Ref.
Pro-inflammatory cytokines (IL-6, TNF-α, IL-1β)	Impair insulin signaling *via* IRS-1 inhibition; Activate hepatic inflammation through NF-κB/JNK pathways	Elevated in T2D, targets for anti-inflammatory therapies	Lara-Guzmán *et al*[48]; Rodrigues *et al*[49]; Liu *et al*[50]; D'Alessandris *et al*[51]
Lipopolysaccharides (LPS) and TLR4-JNK-NF-κB pathway	Trigger hepatic inflammation and insulin resistance following microbial translocation	LPS-driven endotoxemia links dysbiosis to metabolic dysfunction	Li and Wu[58]; Li *et al*[59]; Li *et al*[60]; Tanti and Jager[61]; Li *et al*[62]
FXR and TGR5 receptors	Regulate bile acid metabolism, inhibit gluconeogenesis, promote GLP-1 secretion, and suppress inflammation	Dysregulated in T2D; Therapeutic targets (*e.g.*, FXR agonists)	Grüner and Mattner[39]; Kumari *et al*[43]; Bertolini *et al*[64]; Evangelakos *et al*[65]
Loss of SCFA-producing bacteria (*e.g.*, *Faecalibacterium prausnitzii*)	Reduces gut barrier integrity, lowers SCFA and GLP-1 production, enhances intestinal permeability	Restoration improves insulin sensitivity and gut-liver communication	Garcia-Gutierrez *et al*[66]; He *et al*[67]; Verhoog *et al*[68]; Moran-Ramos *et al*[69]
Expansion of pathobionts (*e.g.*, *Prevotella copri*)	Elevates LPS and BCAA production; activates mTOR-S6K1 pathway impairing insulin signaling	Associated with metabolic endotoxemia, systemic inflammation, and worsened glucose control	Murugesan *et al*[8]; Leite *et al*[70]; Gong *et al*[72]
Microbial metabolites (imidazole propionate, IPA, H_2S, succinate, EE, PAGln)	Modulate insulin signaling, oxidative stress, hepatic lipotoxicity, and inflammatory cascades	Emerging biomarkers and therapeutic targets for metabolic dysfunction	Zeng *et al*[80]; Koh *et al*[81]; Koh *et al*[82]; Cussotto *et al*[83]; Yang *et al*[84]; Munteanu *et al*[85]; Huang *et al*[86]; Xue *et al*[87]; Chen *et al*[88]; Drda and Smith[89]
Bacterial extracellular vesicles	Transfer microbial molecules (*e.g.*, LPS, DNA) across intact barriers to hepatic immune cells, triggering inflammation	Represent a novel barrier-independent mechanism contributing to hepatic insulin resistance	Melo-Marques *et al*[90]; Butcko *et al*[91]
Innate lymphoid cells (ILC3-IL-22 signaling)	Maintain epithelial barrier integrity and mucosal immune balance; Regulate gut homeostasis	ILC dysregulation associated with intestinal permeability defects and systemic inflammation in T2D	Wang *et al*[122]; Yin *et al*[123]; Horn and Sonnenberg[127]
Endocannabinoid system (ECS)	Modulates intestinal permeability, immune activation, hepatic lipid metabolism, and inflammatory tone	Dysregulated ECS signaling contributes to obesity, insulin resistance, and steatohepatitis	Bazwinsky-Wutschke *et al*[133]; Liu *et al*[134]; Cuddihey *et al*[135]; Lipina *et al*[136]; Roser *et al*[137]; O'Sullivan *et al*[138]; Ellermann[139]

IL-1β: Interleukin-1 beta; IL-6: Interleukin-6; TNF-α: Tumor necrosis factor-α; TLR4: Toll-like receptor 4; JNK: C-Jun N-terminal kinase; NF-κB: Nuclear factor kappa B; LPS: Lipopolysaccharide; FXR: Farnesoid X receptor; TGR5: Takeda G protein-coupled receptor 5; SCFAs: Short-chain fatty acids; IPA: Indole-3-propionic acid; H_2S: Hydrogen sulfide; EE: Endogenous ethanol; PAGln: Phenylacetylglutamine; ILC3: Group 3 innate lymphoid cell; ECS: Endocannabinoid system; IRS-1: Insulin receptor substrate-1; GLP-1: Glucagon-like peptide-1; BCAA: Branched-chain amino acids; S6K1: Ribosomal protein S6 kinase 1; mTOR: Mechanistic target of rapamycin; T2D: Type 2 diabetes.

However, central nervous system side effects have limited the clinical utility of systemic CB1 antagonists. Current strategies focus on peripherally restricted CB1 blockers that offer metabolic benefits without neuropsychiatric risks[137,138].

Additionally, gut-derived endocannabinoid-like molecules ("endocannabinoidome") interact with microbial metabolites and bile acid pathways, suggesting a complex tri-directional interplay between microbiota, ECS, and host metabolism[139].

Together, these emerging mechanisms highlight the intricate interplay between gut-derived signals, whether neural (vagal afferents), hormonal (FGF19, FGF21), immune-mediated (ILC3-IL-22 axis), microbial (indole metabolites), or metabolic (endocannabinoid signaling) and hepatic metabolic regulation. Disruption of any component within this network, such as GLP-1 resistance, impaired FGF or IL-22 signaling, loss of beneficial tryptophan metabolites, or dysregulated endocannabinoid activity, can drive hepatic IR, steatosis, and systemic metabolic dysfunction in T2D. Importantly, the emerging recognition of ILCs and the ECS as key modulators of the gut–liver axis expands the therapeutic landscape. Future interventions may combine microbiota-targeted therapies, barrier reinforcement strategies, and molecular modulators of immune and endocannabinoid pathways to synergistically restore gut-liver homeostasis and improve metabolic outcomes in diabetes.

THERAPEUTIC TARGETING OF THE GUT-LIVER AXIS IN DIABETES

Restoring gut-liver axis integrity has emerged as a promising frontier for improving metabolic outcomes in diabetes. Multiple strategies target intestinal barrier reinforcement, immune modulation, and microbiota reshaping. An overview

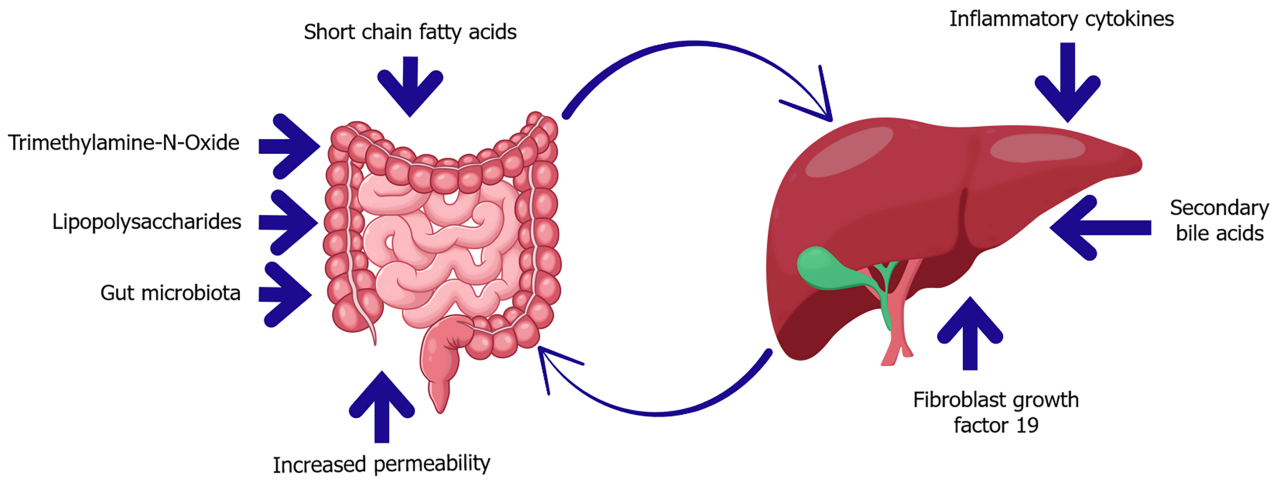

Figure 3 Key mediators of the gut-liver axis in diabetes and insulin resistance. This diagram highlights bidirectional communication between the gut and liver mediated by microbial and host-derived factors. In dysbiosis, microbial metabolites such as trimethylamine-N-oxide and lipopolysaccharides increase gut permeability, enabling translocation of endotoxins into the portal circulation. These signals promote hepatic inflammation and insulin resistance. Conversely, the liver influences gut homeostasis through the secretion of secondary bile acids and fibroblast growth factor 19, which regulate microbial composition and barrier integrity. Short-chain fatty acids also play a protective role, supporting epithelial health and metabolic balance.

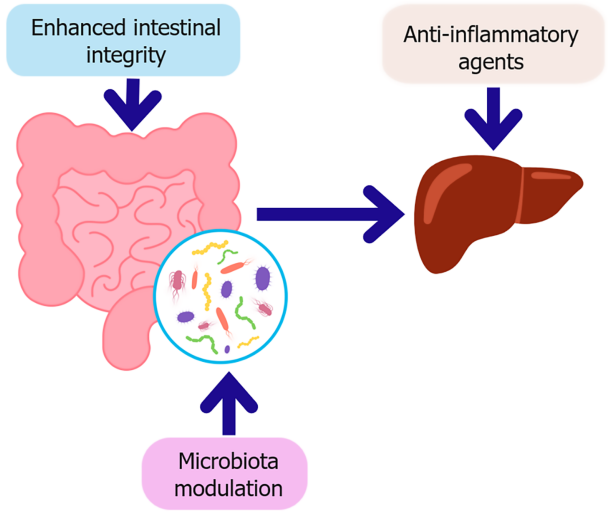

Figure 4 Key therapeutic strategies targeting the gut-liver axis in diabetes. This schematic illustrates therapeutic strategies aimed at restoring gut-liver axis homeostasis in the context of type 2 diabetes. Microbiota modulation, through probiotics, prebiotics, synbiotics, or fecal microbiota transplantation which enhances intestinal integrity by strengthening the epithelial barrier and promoting beneficial metabolites such as short-chain fatty acids. Improved barrier function reduces microbial translocation, subsequently decreasing hepatic inflammation. In parallel, anti-inflammatory agents directly target hepatic immune activation, contributing to improved insulin sensitivity and metabolic regulation.

of current and experimental interventions is provided in Table 2 with a graphical summary provided in Figure 4.

Enhancing intestinal barrier integrity

Reinforcing the intestinal barrier is a central therapeutic goal in diabetes management[140]. Tight junction proteins, claudins, occludin, and zonula occludens-1, seal the paracellular space between enterocytes. Their disruption increases permeability, microbial translocation, and hepatic inflammation[141-143]. Pharmacological interventions, such as zonulin inhibitors (*e.g.*, larazotide acetate) stabilize tight junctions, reduce endotoxemia, and restore barrier function in preclinical models[144-146]. Dietary interventions such as soluble fibers promote SCFA production, particularly butyrate, which strengthens tight junction, while polyphenol-rich diets and omega-3 fatty acids provide antioxidative and anti-inflammatory support to the epithelium[147,148].

Anti-inflammatory interventions

Inflammation plays a pivotal role in gut-liver axis dysfunction. GLP-1 agonists (GLP-1 RAs) and sodium-glucose cotransporter-2 inhibitors (SGLT2i) offer both metabolic and anti-inflammatory benefits[141,149,150]. GLP-1 RAs improve glycemic control, reduce hepatic inflammation, and enhance barrier repair, while SGLT2i therapy decreases C-reactive

Table 2 Key therapeutic strategies targeting gut-liver axis in diabetes

Strategy	Mechanism of action	Examples	Clinical tatus	Ref.
Probiotics	Modulate microbiota composition; Enhance SCFA production; Reinforce gut barrier integrity	*Lactobacillus*, *Bifidobacterium* strains	Approved adjuncts; Variable efficacy	Grylls *et al*[156]; Zhang *et al*[160]; Memon *et al*[162]; McLoughlin *et al*[163]
Prebiotics	Promote growth of beneficial microbes; Increase SCFA levels; Reduce gut permeability and inflammation	Inulin, resistant starch, fructoo-ligosaccharides	Clinically validated for glycemic improvement	McLoughlin *et al*[163]; Luzzi *et al*[165]; Jayedi *et al*[171]
Synbiotics	Synergistic effect of probiotics and prebiotics; Improve glycemia and lipid profiles	Probiotic + fiber combinations	Emerging evidence; Under clinical study	McLoughlin *et al*[163]; Luzzi *et al*[165]; Jayedi *et al*[171]
Postbiotics	Deliver microbial metabolites (*e.g.*, SCFAs) directly to host tissues to modulate metabolism and immunity	SCFA supplements (*e.g.*, acetate, butyrate infusions)	Experimental	McLoughlin *et al*[163]; Fang *et al*[164]; Luzzi *et al*[165]
Fecal microbiota transplantation	Reconstitute healthy microbiome diversity; Restore SCFA and bile acid metabolism	Donor stool capsules or infusions	Experimental; Some success in T2D trials	Wu *et al*[167]; Yadegar *et al*[168]
Zonulin inhibitors	Prevent tight junction disassembly; Restore intestinal barrier integrity	Larazotide acetate (AT-1001)	Phase III for celiac; Early-stage for T2D	Choi *et al*[73]; Górecka *et al*[143]; Yonker *et al*[145]; Tajik *et al*[146]; Jayashree *et al*[178]; Yuan *et al*[179]
Dietary interventions	Enrich SCFA-producing bacteria; Upregulate tight junction proteins; Reduce systemic inflammation	High-fiber and polyphenol-rich diets (berries, teas)	Clinically recommended adjunct therapy	Verhoog *et al*[68]; Mazhar *et al*[147]; Han *et al*[169]
GLP-1 receptor agonists	Enhance insulin secretion; Reduce hepatic and gut inflam-mation; Improve barrier function	Liraglutide, semaglutide	Approved for T2D and obesity	Zhang *et al*[141]; Alharbi[149]
SGLT2 inhibitors	Improve glycemic control; Reduce systemic and hepatic inflammation	Empagliflozin, dapagliflozin	Approved for T2D and cardiovascular protection	Theofilis *et al*[150]; Zhang *et al*[151]
FXR agonists	Regulate bile acid metabolism; Restore barrier function; Suppress liver fibrosis and inflammation	Obeticholic acid	Approved for PBC; Under investigation for NASH	Zhang *et al*[152]
TLR4 antagonists	Block LPS signaling to prevent endotoxin-driven inflam-mation	Eritoran	Experimental	Liang *et al*[170]
Cytokine inhibitors	Suppress pro-inflammatory cytokines (*e.g.*, IL-1β) to reduce hepatic and systemic inflam-mation	Canakinumab, anakinra	Under investigation	Everett *et al*[157]; Howard *et al*[158]
AhR agonists	Activate anti-inflammatory pathways; Stabilize tight junctions	Indole derivatives (*e.g.*, FICZ, 5-HIAA)	Experimental; Preclinical promising	Cussotto *et al*[83]; Zheng *et al*[120]; Du *et al*[121]; Pernomian *et al*[159]
Gut-targeted biologics/probiotics	Modulate mucosal immunity; Reduce pro-inflammatory responses locally	Engineered probiotics, oral cytokine blockers	Preclinical and early-phase trials	Zhang *et al*[160]

This table summarizes current and emerging interventions aimed at modulating the gut microbiota, enhancing intestinal barrier integrity, reducing inflammation, and restoring gut-liver metabolic signaling. GLP-1: Glucagon-like peptide-1; SCFAs: Short-chain fatty acids; SGLT2: Sodium-glucose cotransporter-2; FXR: Farnesoid X receptor; TLR4: Toll-like receptor 4; AhR: Aryl hydrocarbon receptor; T2D: Type 2 diabetes; LPS: Lipopolysaccharide; PBC: Primary biliary cirrhosis; NASH: Non-alcoholic steatohepatitis; IL-1β: Interleukin-1 beta; FICZ: 6-formylindolo[3,2-b]carbazole; 5-HIAA: 5-hydroxyindole-3-acetic acid.

protein and interleukin-6[151]. Furthermore, FXR agonists, such as obeticholic acid, modulate bile acid metabolism, improve intestinal integrity, and suppress liver fibrosis[152,153].

Bile acid sequestrants (BASs) such as colesevelam and colestimide also modulate enterohepatic signaling. A systematic review and meta-analysis of 17 randomized controlled trials involving 2950 patients showed significant glycated hemoglobin (HbA1c) reductions. These effects are attributed to delayed bile acids reabsorption, enhanced GLP-1

secretion, and FXR/TGR5 modulation. Though long-term safety data are limited, BASs are recommended as adjunctive therapy in T2D[154].

Emerging therapies include TLR4 antagonists, cytokine inhibitors such as canakinumab targeting IL-1β, and AhR agonists derived from microbial tryptophan metabolites, which enhance mucosal immunity and barrier function[155-159]. Engineered probiotics and oral anti-cytokine therapies are under investigation to restore mucosal balance without systemic immunosuppression[160,161].

Microbiota modulation

Modulating gut microbiota is another therapeutic cornerstone. Probiotics, particularly *Lactobacillus* and *Bifidobacterium* demonstrate modest improvements in glycemic and lipid profiles, especially with metformin[162]. Prebiotics such as inulin and resistant starch support SCFA-producing bacteria, improving barrier function and reducing inflammation [163]. Synbiotics, which combine probiotics and prebiotics, demonstrate synergistic benefits, while postbiotics (direct delivery of SCFAs) offer a novel therapeutic approach[164-166].

Fecal microbiota transplantation (FMT) enhances microbial diversity and insulin sensitivity, although donor variability and standardization challenges remain[167,168]. Additionally, high-fiber and polyphenol-rich diets effectively modulate the microbiome, increase SCFA production, and strengthen barrier function, contributing to improved metabolic outcomes[68,147,169].

LIMITATIONS OF CURRENT EVIDENCE AND KNOWLEDGE GAPS

Despite extensive research on the gut-liver axis in T2D, several limitations hinder the translation of mechanistic insights into clinically effective therapies. One major challenge is the gap between preclinical and clinical findings. While interventions such as FXR agonists, TLR4 antagonists, and indole-derived AhR ligands demonstrate robust metabolic and anti-inflammatory effects in rodent models, human trials often yield inconsistent, modest, or inconclusive results. For instance, the TLR4 antagonists eritoran failed to show significant clinical efficacy in metabolic or inflammatory conditions, possibly due to species-specific differences in innate immune pathways, genetic polymorphisms in TLR genes, or differences in gut microbial composition[170].

Moreover, while probiotic and prebiotic therapies show promise in experimental models, their outcomes in humans remain variable. Meta-analyses often reveal only modest and statistically insignificant changes in HbA1c or insulin sensitivity. Efficacy appears to depend on multiple factors, including strain selection, dosage, treatment duration, and baseline host microbiota profiles[171]. This variability reflects the complexity of host-microbe interactions and the need for personalized therapeutic approaches.

Furthermore, many mechanistic links remain correlative rather than causative. For example, alterations in microbial composition or metabolite levels such as ImP or SCFAs are frequently observed in T2D, but whether these changes are drivers of disease or markers of metabolic dysregulation remain unclear[172].

Similarly, while preclinical data support roles of microbial EVs, endocannabinoid signaling, and mucosal immune mediators such as IL-22 in regulating metabolic and inflammatory pathways, their specific functions in human T2D and gut-liver axis disorders are not well established. The mechanisms by which microbial EVs influence hepatic immunity, the context-dependent effects of cannabinoid receptor, and the dual roles of IL-22 still require validation in human studies and clinical trials[2,133,173,174].

A further challenge is the lack of longitudinal studies, large-scale trials, and standardized methodologies. Many existing studies are limited by small sample sizes and lack of stratification by key variables such as microbiota composition, diet, genetics, and comorbidities, complicating interpretation and reproducibility[175].

In summary, while the gut-liver axis presents substantial therapeutic potential, advancing the field requires moving beyond associative evidence to establish causality and therapeutic relevance through rigorous, human-centered research. Addressing these limitations is essential for developing safe, effective, and personalized interventions in T2D management.

CHALLENGES AND FUTURE DIRECTIONS

Despite advances in understanding the gut-liver axis, several challenges continue to impede the clinical translation of related therapies. One significant barrier is the high degree of microbial heterogeneity among individuals, which complicates therapeutic standardization and highlights the need for precision medicine approaches[176].

A major limitation is the lack of validated biomarkers to stratify patients and monitor therapeutic responses[177]. While serum markers such as LPS, zonulin, and ImP, are under investigation, they require further validation before being adopted in routine clinical practice[178,179]. Furthermore, regulatory hurdles, concerns over long-term safety, and complexities in manufacturing limit the clinical use of advanced interventions like FMT and engineered probiotics[180,181].

Future strategies should integrate multi-omics profiling, including microbiome, metabolome, transcriptome, and host genomic data, to build individualized therapeutic models. These datasets can inform stratified clinical trials and enable biomarker-driven intervention tailored to specific patient subtypes[182,183]. Moreover, novel tools such as gut organoids, humanized microbiota animal models, and artificial intelligence hold promise for bridging the translational gap between experimental studies and human applications[184].

Emerging insights into the gut-liver-brain axis also indicate that interventions targeting the gut may offer benefits beyond metabolic regulation, potentially improving neurocognitive outcomes in diabetes. Integrating metabolic and neurocognitive strategies could lead to more holistic approaches in managing T2D[185-187].

Together, these challenges and opportunities highlight a pivotal turning point in gut-liver research. To fully realize the therapeutic potential of this axis, future efforts must balance mechanistic complexity with clinical precision and personalized care models.

PROPOSED INTEGRATIVE FRAMEWORK FOR GUT–LIVER CROSSTALK IN T2D

To advance conceptual clarity and support translational progress, this review proposes an integrative framework that synthesizes microbial, immune, metabolic, and neuroendocrine pathways implicated in the gut-liver crosstalk in the pathogenesis of T2D. This model maps the convergence of dysbiotic microbial signals, inflammatory mediators, hormonal feedback loops, and neural circuits to explain how disruptions across this network contribute to hepatic IR and systemic metabolic dysfunction.

The framework also informs therapeutic innovation by aligning key pathogenic mechanisms with corresponding intervention strategies. These include microbial modulation through probiotics and FMT, intestinal barrier reinforcement using dietary fibers, SCFAs and zonulin inhibitors, immune regulation *via* agents targeting IL-22 and AhR signaling, and restoration of enterohepatic signaling *via* GLP-1 receptor agonists and FXR/TGR5 modulators. By integrating mechanistic insight with intervention targets, the model supports a precision medicine approach aimed at tailoring therapies to individual microbial, immune, and metabolic profiles. The proposed conceptual framework is visually represented in Figure 5.

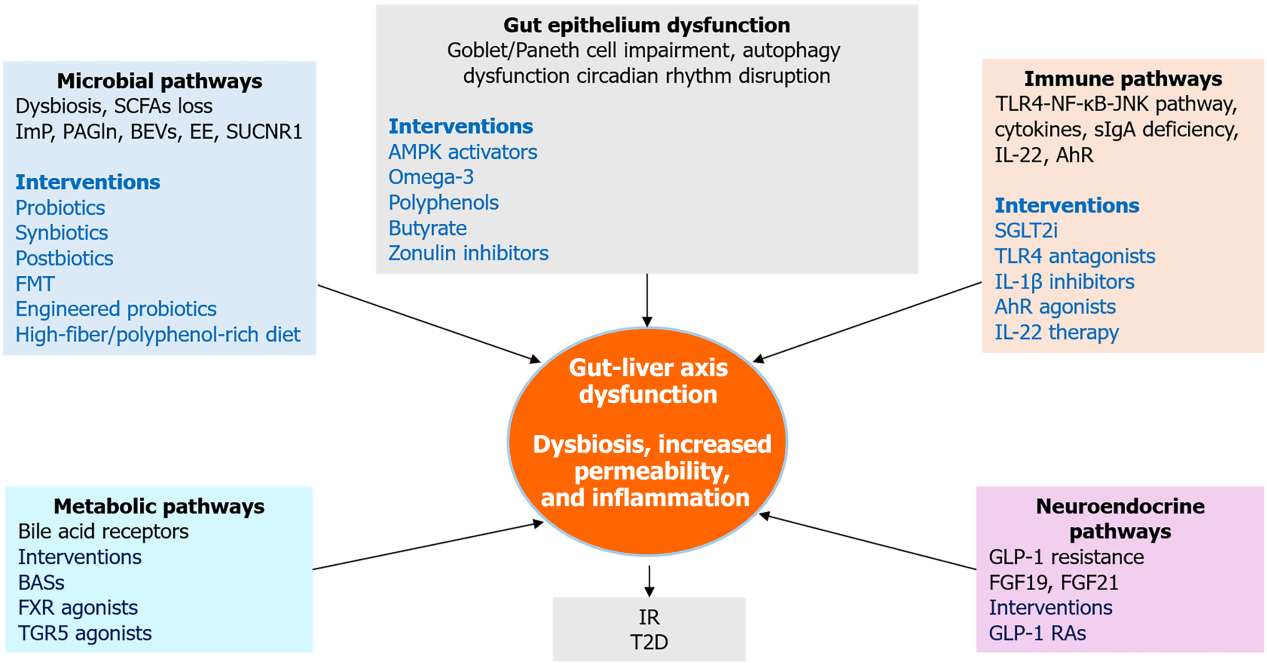

Figure 5 Integrative therapeutic and mechanistic pathways linking gut and liver in type 2 diabetes. This integrative framework illustrates how five key mechanistic domains, microbial dysbiosis, gut epithelial dysfunction, immune activation, neuroendocrine disruption, and metabolic signaling which converge to drive gut-liver axis dysfunction in type 2 diabetes. Each domain includes representative molecular mediators and therapeutic interventions. The central node highlights dysbiosis, increased permeability, and inflammation as core features linking upstream disruptions to downstream insulin resistance and hepatic steatosis. SCFAs: Short-chain fatty acids; ImP: Imidazole propionate; PAGln: Phenylacetylglutamine; BEVs: Bacterial extracellular vesicles; EE: Endogenous ethanol; FMT: Fecal microbiota transplantation; BASs: Bile acid sequestrants; FXR: Farnesoid X receptor; TGR5: Takeda G protein-coupled receptor 5; AMPK: Adenosine 5'-monophosphate-activated protein kinase; IR: Insulin resistance; T2D: Type 2 diabetes; TLR4: Toll-like receptor 4; JNK: C-Jun N-terminal kinase; NF-κB: Nuclear factor kappa B; sIgA: Secretory immunoglobulin A; IL-1β: Interleukin-1 beta; IL-22: Interleukin-22; AhR: Aryl hydrocarbon receptor; SGLT2i: Sodium-glucose cotransporter-2 inhibitors; FGF19: Fibroblast growth factor 19; FGF21: Fibroblast growth factor 21; GLP-1: Glucagon-like peptide-1; GLP-1 RAs: Glucagon-like peptide-1 receptor agonists.

CONCLUSION

The gut-liver axis plays a central role in the development and progression of T2D by integrating microbiota, immune, barrier, and metabolic signals. Disruption of this axis contributes to IR, hepatic steatosis, and systemic metabolic dysfunction. Therapeutic strategies that aim to restore gut-liver homeostasis through microbiota modulation, enhancement of barrier integrity, anti-inflammatory interventions, and regulation of gut-derived signaling pathways

offer promising opportunities to improve clinical outcomes. However, realizing this potential requires a personalized approach that incorporates individual microbial, immune, and metabolic profiles. Advances in microbiome research, multi-omics profiling, and precision medicine tools are rapidly expanding this therapeutic landscape. Despite such progress, several challenges remain, including the lack of validated biomarkers, variability in treatment responses, and limited long-term safety data. Furthermore, a deeper understanding of the gut-liver-brain network may help address complications beyond metabolism, including cognitive decline. Moving forward, combining gut-liver axis therapies with existing treatments may support a more holistic, durable, and individualized approach to diabetes management.

FOOTNOTES

Author contributions: Abdalla MMI performed the research and wrote the paper.

Conflict-of-interest statement: The author has no conflict of interest to declare.

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